This has been an effective answer to the criticism that physicians are doing nothing about the problem. Newspaper advertising has appeared in 450 newspapers in 34 counties. A California Medical Association sponsored radio program, "California Caravan," broadcast weekly over Mutual Network discussing the merits of voluntary prepaid health insurance, has had a listening audience of half a million people.

California Medical Association's aggressive, militant program has been a vital factor in a successful attack against political medicine. However, we must give credit to other factors which have aided us, among them the swing of the pendulum against regimentation and public controls as reflected in our recent congressional election trends and favorable economic trends. These trends may not continue indefinitely and if economic lags and depressions come along, we may expect sharp recurrences of demands for political medical cure-alls.

Public education must be continued on a long term basis, and if our objective of providing proper medical care on a voluntary basis in this country is gained I believe that we should recommend:

1. Capitalizing on our tremendous gains made in voluntary health insurance coverage with a goal of at least one million additional members during 1947, or in two years a goal of four to six million, which would approximate the number covered under the proposed compulsory systems.

2. Broadening of newspaper and radio advertising campaigns to inform every California citizen of the availability and merits of voluntary plans.

3. Continuation of the "Voluntary Health Insurance Weeks" so as to clinch the sales in all local communities.

4. Extension of aid to all County Medical Societies in establishing a sound public relations program, striving to inform the citizens of the state about the part that local physicians play in meeting community problems and what they are doing to take the economic shock out of illnesses.

The physicians of California, to do these things, must be vigilant and prepared. They must assume their responsibilities as citizens and physicians looking toward community betterment and improved health under a system which has made this the healthiest and greatest nation in the world.

The Increasing Importance of Q Fever Infection*

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QUEENSLAND fever, or Q fever, as it has come to be known, has been considered a rare and relatively remote rickettsial infection, obscure as to the mode of contagion and bizarre in its clinical manifestations. The total number of cases which have been studied is less than 200, nearly all in Australia, and they may be classified in four general categories. The first of these is the series of cases in Queensland, 176 in number, discussed as a group by Derrick in 1942.[8] The second is the small group of five infections, two in ticks and three in patients, observed in Mantana between 1938 and 1941 and reported rather incompletely in three separate articles.[6,10,12] The third is the laboratory outbreak in 15 patients, one of whom died, at the Institute of National Health, Bethesda, Maryland.[11,13] And the fourth category includes a series of cases in the Western United States diagnosed and reported solely on the basis of positive serological studies on sick or convalescent individuals.[7]

Up until last year (1945), our total knowledge of the clinical manifestations of the disease, Q fever, was based on these four sources, so that it may readily be seen that the average physician in the United States would have had no direct concern with the disease and might well have considered it just one more medical curiosity. Such an opinion would have been supported by the fact that certain well established characteristics of rickettsial diseases, such as occur in various forms of typhus, and in the Rocky Mountain Spotted Fever (R.M.S.F.G.) group of tick-borne infections do not occur in Q fever. The cardinal differences may best be shown as follows:

	Typhus and R.M.S.F.G.	Q Fever
1. Rash	Common	Very rare
2. Leucocytosis	Common	Uncommon
3. Weil Felix Test	Positive	Negative
4. Extra cellular forms of rickettsiae	None	Common
5. Filterable form of rickettsiae	None	Occur
6. Mortality	May be high	Low (about 2%)

IDENTIFIED IN NEW LOCALITIES IN 1945

However, in 1945, Q fever passed from a limited orbit of concern to the broad sphere of world-wide interest when it was identified in three new localities: the Panama Canal Zone, Italy and the Balkans.[1*] In each instance the presence of Q fever infection was not believed to be due to a chance importation. Rather, it was considered as due to an endemic focus, long present, but only just brought to light by the increase in diagnostic

* Read before the Section on Public Health at the Seventy-fifth Annual Session of the California Medical Association, Hotel Biltmore, Los Angeles, May 7-10, 1946.

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* Subsequent to the presentation of this paper, Q fever was reported in Texas in the Public Health Reports for May 31, 1946. Vol. 61, p. 784.

facilities contingent upon World War II medical conditions. As the writer is familiar, first-hand, with the identification of the infection in Panama,[1] which was the first area discovered in Latin America, the exact circumstances which led to proving the presence of the disease should be briefly considered.

Of first importance was the fact that a tropical fever, clinically resembling a rickettsial infection of the Q fever type, had been recognized in this area for years but adequate diagnostic studies necessary for its detection had never been carried out. Of equal importance was the fact that primary atypical bronchopneumonia or virus pneumonitis of unknown etiology occurred commonly, and consequently offered an excellent opportunity to study the cause of this baffling problem in the tropics. In a diagnostic survey of this condition which included guinea pig inoculation studies, the first case of Q fever was encountered in a soldier who had not been out of the Canal Zone in many months. Not only were the rickettsia recovered after animal inoculation and a strongly positive complement fixation test secured on the patient's blood serum, but subsequent immunization and neutralization tests carried out at the National Institute of Health at Bethesda, Maryland, proved the identity of the Panamanian strain of Q fever rickettsia and the American strain in the United States. The splendid collaboration of the United States Public Health Service in this work greatly facilitated the complete diagnostic studies necessary to establish beyond a doubt the presence of Q fever in Central America.

With the establishment of new and widely separated foci of Q fever infection, its increasing importance to the medical profession is obvious. It seems certain that the presence of this disease will soon be detected in other new localities as our knowledge concerning it grows and as modern diagnostic tests are applied more widely. Because of the mounting interest in Q fever, a review of the little-known facts in regard to its origin and transmission are of paramount importance today.

HISTORICAL

The first published accounts on Q fever came out just ten years ago when Derrick reported the clinical findings in nine patients, all of whom were slaughter-house workers,[9] and Burnet and Freeman reported their laboratory studies showing that the infection was rickettsial in origin.[2] They demonstrated the febrile response of guinea pigs following the injection of patients' blood, the development of a specific immunity in these guinea pigs, and the presence of rickettsia in the livers and spleens of mice inoculated with infected guinea pig tissue although the infection transmitted to mice was unapparent clinically. The next year, 1938, specific serum agglutination tests were reported by the same authors in the *Medical Journal of Australia*.[3] This same journal contained a series of articles on Q fever in 1939. A number of articles also appeared on this subject in the *Australian Journal of Experimental Biology and Medical Science* between 1938 and 1942. The name *Rickettsia burneti* was accepted for the specific type of rickettsia causing Q fever infection.

In December, 1938, Cox reported the identification of Q fever infection in ticks in Montana.[6] This infection occurred in a filterable virus form. At the same time Dyer described the first patient in the United States (Montana) with the disease, Q fever.[10] In 1940 the laboratory outbreak of Q fever at the National Institute of Health was described in the U. S. Public Health Reports. Fifteen individuals were affected and all had pneumonitis. It was noted that the laboratory workers who contracted the disease were in different parts of the building and some were not even in direct contact with the experimental studies which were in progress. Since then, the same journal has carried a series of articles dealing with the cultural characteristics, immunology and epidemiology of the disease. In the last five years a few articles have appeared in other medical journals, including a report of two additional American cases occurring in Montana in 1941.[12]

Q FEVER INFECTION IN ANIMALS

In seeking an animal source of infection, it was soon found that a small bush animal in Queensland, the bandicoot, *Iscodon torosus*, served as a reservoir for *Rickettsia burneti*. On Moreton Island 34 per cent of the bandicoots showed positive serum agglutination tests for Q fever. Rickettsia were recovered from ticks removed from bandicoots. Three other marsupials and seven species of rodents proved susceptible to laboratory infection. Two species of rats showed positive serum agglutination tests.[8]

The demonstration that approximately 1.5 per cent of 984 cattle tested in Queensland showed positive serum agglutination tests for Q fever, and that calves were susceptible to laboratory infection proved of great epidemiological significance; because, aside from laboratory workers, nearly all cases of human infection have been described in either slaughter house or dairy workers whose occupation brought them in contact with cattle, alive or dead.[8]

Q FEVER INFECTION IN TICKS

A tick, *Haemaphysalis humerosa*, is commonly found on bandicoots and probably serves as a natural host for *Rickettsia burneti*, as it may be found in large numbers in the epithelial cells lining the intestinal canal and in the intestinal lumen of these animal parasites. These ticks could readily be infected in the laboratory in every instance when they were allowed to feed on guinea pigs previously inoculated with Q fever, and they can in all stages transmit this infection to guinea pigs. Hereditary transmission in these ticks has not been proven. *Haemaphysalis humerosa* attacks small animals only and is found on other animals than the bandicoot, including the opossum. Another tick, the scrub tick, *Ixodes holocychus*, is also an ectoparasite of the bandicoot and is a host for Q fever rickettsia. However, unlike Haemaphysalis, it attacks not only small animals, but also cattle and

man. At least three other ticks parasitic to small animals may harbor *Rickettsia burneti.*

Also, a common cattle tick, *Boophilis annulatus,* can take up Q fever rickettsia from calves and retain them for months. They are present in its feces, where they have been shown to remain infective for as long as 87 days. Two other common cattle ticks can also act as vectors, and a kangaroo tick is known to be susceptible to laboratory infection.

PRESENT CONCEPT OF HUMAN INFECTION

In Queensland it is evident that the bandicoot acts as a reservoir for Q fever infection and that a tick acts as a vector to complete the basic cycle of infection from bandicoot to bandicoot. Another tick acts as a vector between bandicoots and cattle and probably man. In only a few instances has a history of tick bites preceded the development of Q fever in humans. It is presumed that an individual is directly infected by the tick only when feces are first dropped on the skin and the tick then breaks the skin through the fecal deposit, thereby introducing rickettsia into the blood stream.

It is commonly believed at present, although not proven, that most humans are infected by dry tick feces containing viable rickettsia and that this material is inhaled as dust. It might cause infection by contact with an abrasion in the skin. The portal of entry by inhalation would best explain the widespread but irregular infection in the abattoirs and the repeated laboratory outbreaks which have occurred, often involving personnel not directly connected with the experimental work. A similar mode of infection may also be responsible for certain virus infections, such as ornithosis.

DIAGNOSTIC STUDIES

Accurate diagnosis of Q fever has heretofore been retarded for certain very definite reasons. First, the epidemiological and clinical features of the disease have not been widely known among practicing physicians and consequently satisfactory diagnostic criteria have not been applied. Second, animal inoculation studies to determine the etiology of the disease are laborious and require technical skill which is frequently not available. Third, because the antigen for serum agglutination tests and complement fixation tests has not been available for general use, these tests have not been practical clinical procedures. Fourth, laboratories in Australia and the United States which have carried out experimental and diagnostic studies dealing with Q fever have had serious laboratory infections, incapacitating their personnel, greatly handicapping the studies.

Although a definite diagnosis of Q fever cannot be made from the clinical findings alone, the presence of this infection may be suspected in cases of atypical bronchopneumonia (virus pneumonitis) of unknown etiology and similar febrile illnesses unaccompanied by pneumonitis.[5] At present, unless facilities for guinea pig inoculation studies are available, a positive diagnosis of Q fever infection can only be made from serological studies. The results of such studies will usually come too late to be of value during the course of the acute illness, which usually lasts seven to ten days, as the agglutination and complement fixation tests are not clearly positive until the second week after onset of the disease. However, serum may be forwarded to the National Institute of Health at Bethesda, Maryland, for these serological tests, and positive results will establish the identity of the infection.

SUMMARY

1. The incidence, certain historical aspects and the epidemiology of Q fever infection have been reviewed.

2. The presence of the etiological agent, *Rickettsia burneti,* which causes Q fever has recently been reported from Panama, Italy, the Balkans and Texas where it has caused "virus pneumonitis" infections.

3. As Q fever is now known to be widely spread throughout the world, and as serological tests satisfactory for diagnosis have been developed, this infection assumes an increase in clinical and public health importance since its discovery ten years ago.

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